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L5   ANSWER 1 OF 14  CA  COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER:        143:223605  CA
TITLE:                   RNA interference-mediated inhibition of EGFR
                         (epidermal growth factor receptor) gene expression
                         using siNA's (short interfering nucleic acids)
INVENTOR(S):             McSwiggen, James; Beigelman, Leonid; Pavco, Pamela;
                         Fosnaugh, Kathy; Jamison, Sharon
PATENT ASSIGNEE(S):      Sirna Therapeutics, Inc., USA
SOURCE:                  U.S. Pat. Appl. Publ., 368 pp., Cont.-in-part of Appl.
                         No. PCT/US04/016390.
                         CODEN: USXXCO
DOCUMENT TYPE:           Patent
LANGUAGE:                English
FAMILY ACC. NUM. COUNT:  233
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US 2003-422704 A2 20030424
US 2003-652791 A2 20030829

AB This invention relates to compds. and methods useful for modulating EGFR gene (e.g., HER1, **HER2**, HER3, and/or HER4) expression by **RNAi** using siNA's. The siNA's may include **siRNA** and modified **siRNA**, double-stranded RNA, micro-RNA, and short hairpin RNA mols.. The siNA's may be used in the treatment of cancer. Thus, chemical modified **siRNA**'s significantly reduced HER1 and **HER2** gene expression in vitro in A549 (ovarian cancer) cells.

L5 ANSWER 2 OF 14 CA COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 142:386024 CA
TITLE: Nucleic acid treatment of diseases or conditions related to expression levels of RAS, **HER2** and HIV genes
INVENTOR(S): McSwiggen, James
PATENT ASSIGNEE(S): Sirna Therapeutics, Inc., USA
SOURCE: U.S. Pat. Appl. Publ., 143 pp., Cont.-in-part of U.S. Ser. No. 693,059.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 233
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AB The present invention relates to nucleic acid mols., including enzymic nucleic acid mols., that modulate the expression of Ras genes (such as K-RAS, H-RAS, and/or N-RAS), **HER2** gene, and HIV genes. Gene sequences of human c-Ki-ras2, c-Ha-ras1, and **HER2** genes and HIV-1 genes were screened for accessible sites using a computer-folding algorithm. DNAzymes are designed for c-Ki-ras2, c-Ha-ras1, and **HER2**; for modulation of HIV-1 genes, the present invention provides DNAzymes, hammerhead ribozymes, Inozymes, Zinzymes, Amberzymes, and other enzymic nucleic acids.

L5 ANSWER 3 OF 14 CA COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 142:183225 CA
TITLE: RNA interference-mediated inhibition of gene expression using chemically modified short interfering nucleic acids
INVENTOR(S): McSwiggen, James; Chowrira, Bharat; Beigelman, Leonid; Macejak, Dennis; Zinnen, Shawn; Pavco, Pamela; Haeberli, Peter; Morissey, David; Fosnaugh, Kathy; Jamison, Sharon; Usman, Nassim; Thompson, James; Vargeese, Chandra; Wang, Weimen; Chen, Tonqian; Vaish, Narendra
PATENT ASSIGNEE(S): Sirna Therapeutics, Inc., USA
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AB The present invention concerns methods and reagents useful in modulating gene expression in a variety of applications, including use in therapeutic, diagnostic, target validation, and genomic discovery applications. Specifically, the invention relates to synthetic chemical modified small nucleic acid mols., such as short interfering nucleic acid (siNA), short interfering RNA (**siRNA**), double-stranded RNA (**dsRNA**), micro-RNA (miRNA), and short hairpin RNA (shRNA) mols. capable of mediating RNA interference (**RNAi**) against target nucleic acid sequences. Introduction of chemical modified nucleotides into nucleic acid mols. provides a powerful tool in overcoming potential limitations of in vivo stability and bioavailability inherent to native RNA mols. Unlike native unmodified **siRNA**, chemical modified siNA can also minimize the possibility of activating interferon activity in humans. Modifications are described including pyrimidine or purine nucleotides with 2'-deoxy-2'-fluoro or 2'-O-Me groups, phosphorothioate backbone modification, terminal residues comprising inverted deoxy thymidine or inverted deoxy abasic moieties, linking the sense and antisense strands with glyceryl succinate or dodecanoic acid or other linkers, and conjugation of targeting ligands (N-acetylgalactosamine, pteroic acid, peptides, or phospholipids) to the oligonucleotide termini. Thus, the serum stability of siNA constructs consisting of all RNA nucleotides containing two thymidine nucleotide overhangs have a half-life in human serum of 15 s, whereas chemical modified siNA constructs remained stable in serum for 1 to 3 days depending on the extent of modification. The small nucleic acid mols. are useful in the treatment of any disease or condition that responds to modulation of gene expression or activity in a cell, tissue, or organism. Three nuclease-resistant siNA mols. targeting site 1580 of hepatitis B virus RNA are designed using Stab 7/8 chemical and a 5'-terminal conjugate moiety (a branched cholesterol conjugate, a branched phospholipid conjugate, and a polyethylene glycol conjugate) showed significant stability in human and mouse serum (t1/2 = 10-408 h) and human liver extract (t1/2 = 28-43 h); the most stable siNA with all purine positions in the antisense strand with 2'-O-Me nucleotides had a half-life of 816 h in human liver extract

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ACCESSION NUMBER: 141:319980 CA
TITLE: RNA interference-mediated inhibition of gene expression using chemically modified short interfering nucleic acids
INVENTOR(S): Mcswiggen, James; Chowrira, Bharat; Beigelman, Leonid; Macejak, Dennis; Zinnen, Shawn; Pavco, Pamela; Haeberli, Peter; Morrissey, David; Fosnaugh, Kathy; Jamison, Sharon; Usman, Nassim; Thompson, James;

	Vargeese, Chandra; Wang, Weimin; Chen, Tongqian; Vaish, Narendra
PATENT ASSIGNEE(S):	USA
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	CODEN: USXXCO
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AB The present invention concerns methods and reagents useful in modulating gene expression in a variety of applications, including use in therapeutic, diagnostic, target validation, and genomic discovery applications. Specifically, the invention relates to synthetic chemical modified small nucleic acid mols., such as short interfering nucleic acid (siNA), short interfering RNA (**siRNA**), double-stranded RNA (**dsRNA**), micro-RNA (miRNA), and short hairpin RNA (shRNA) mols. capable of mediating RNA interference (**RNAi**) against target nucleic acid sequences. Introduction of chemical modified nucleotides into nucleic acid mols. provides a powerful tool in overcoming potential limitations of in vivo stability and bioavailability inherent to native RNA mols. Unlike native unmodified **siRNA**, chemical modified siNA can also minimize the possibility of activating interferon activity in humans. Modifications are described including pyrimidine or purine nucleotides with 2'-deoxy-2'-fluoro or 2'-O-Me groups, phosphorothioate backbone modification, terminal residues comprising inverted deoxy thymidine or inverted deoxy abasic moieties, linking the sense and antisense strands with glyceryl succinate or dodecanoic acid or other linkers, and conjugation of targeting ligands (N-acetylgalactosamine, pteroic acid, peptides, or phospholipids) to the oligonucleotide termini. Thus, the serum stability of siNA constructs consisting of all RNA nucleotides containing two thymidine nucleotide overhangs have a half-life in human serum of 15 s, whereas chemical modified siNA constructs remained stable in serum for 1 to 3 days depending on the extent of modification. The small nucleic acid mols. are useful in the treatment of any disease or

condition that responds to modulation of gene expression or activity in a cell, tissue, or organism. Three nuclease-resistant siNA mols. targeting site 1580 of hepatitis B virus RNA are designed using Stab 7/8 chemical and a 5'-terminal conjugate moiety (a branched cholesterol conjugate, a branched phospholipid conjugate, and a polyethylene glycol conjugate) showed significant stability in human and mouse serum (t1/2 = 10-408 h) and human liver extract (t1/2 = 28-43 h); the most stable siNA with all purine positions in the antisense strand with 2'-O-Me nucleotides had a half-life of 816 h in human liver extract

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ACCESSION NUMBER: 139:240342 CA
TITLE: RNA interference mediated inhibition of gene expression using chemically modified short interfering nucleic acid
INVENTOR(S): Mcswiggen, James; Beigelman, Leonid; Chowrira, Bharat; Pavco, Pamela; Fosnaugh, Kathy; Jamison, Sharon; Usman, Nassim; Thompson, James
PATENT ASSIGNEE(S): Sirna Therapeutics, Inc., USA
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AB The present invention concerns methods and reagents useful in modulating gene expression in a variety of applications, including use in therapeutic, diagnostic, target validation, and genomic discovery applications. Specifically, the invention relates to small nucleic acid mols., such as short interfering nucleic acid (siNA), short interfering RNA (**siRNA**), double-stranded RNA (**dsRNA**), micro-RNA (miRNA), and short hairpin RNA (shRNA) mols. capable of mediating RNA interference (**RNAi**) against target nucleic acid sequences. Exemplary siNA mols. are synthesized in tandem using standard phosphoramidite synthesis chemical and a cleavable linker, for example a succinyl-based linker, followed by a one-step purification process that provides **RNAi** mols. in high yield. Chemical modifications (2'-O-Me and 2'-deoxy-2'-fluoro groups, phosphorothioate linkages, 5'-terminal caps comprising an inverted deoxy abasic moiety, etc.) in siNA constructs are selected to yield nuclease resistance while preserving the ability to mediate **RNAi** activity. The siNA mols. are designed that can bind to target mRNAs for vascular endothelial growth factor receptors, BCL2, **HER2/neu**/ c-Myc, PCNA, RELA, PTP1B, BACE, CHK1, PKC-α, and EGFR/HER1, and are optionally individually analyzed by a computer folding algorithm to assess whether the siNA mol. can interact with the target sequence. The siNA mols. are useful in the treatment and diagnosis of any condition that responds to modulation of gene expression or activity in a cell, tissue, or organism.

L5 ANSWER 6 OF 14 CA COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 139:191417 CA
TITLE: RNA interference-mediated inhibition of epidermal growth factor receptor gene expression using short interfering nucleic acids
INVENTOR(S): McSwiggen, James; Pavco, Pamela; Beigelman, Leonid; Fosnaugh, Kathy; Jamison, Sharon

PATENT ASSIGNEE(S): Ribozyme Pharmaceuticals, Incorporated, USA; Sirna Therapeutics, Inc.
SOURCE: PCT Int. Appl., 171 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 233
PATENT INFORMATION:

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AB The present invention concerns methods and reagents useful in modulating epidermal growth factor receptor gene (HER1, **HER2** also known as **erbB2/neu**, HER3, and HER4) expression in a variety of applications, including use in therapeutic, diagnostic, target validation, and genomic discovery applications. Specifically, the invention relates to small nucleic acid mols., such as short interfering nucleic acid (siNA), short interfering RNA (**siRNA**), double-stranded RNA (**dsRNA**), micro-RNA (miRNA), and short hairpin RNA (shRNA) mols. capable of mediating RNA interference (**RNAi**) against epidermal growth factor receptor gene expression and/or activity. Exemplary siNA mols. are synthesized in tandem using standard phosphoramidite synthesis chemical and a cleavable linker, for example a succinyl-based linker, followed by a one-step purification process that provides **RNAi** mols. in high yield. Chemical modifications (2'-O-Me and 2'-deoxy-2'-fluoro groups, phosphorothioate linkages, 5'-terminal caps comprising an inverted deoxy abasic moiety, etc.) in siNA constructs are selected to yield nuclease resistance while preserving the ability to mediate **RNAi** activity. The siNA mols. are designed that can bind to each target and are optionally individually analyzed by a computer folding algorithm to assess whether the siNA mol. can interact with the target sequence. The efficacy of siNAs targeting epidermal growth factor receptor genes are tested in cell culture using, for example, SKBR-3 or SKOV-3 cells, to detn the extent of RNA and protein inhibition; after an optimal transfection agent concentration is chosen, a RNA time-course of inhibition is performed with the lead siNA mol(s). The siNA mols. are useful in the treatment and diagnosis of cancer.

L5 ANSWER 7 OF 14 CA COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 139:240335 CA
TITLE: RNA interference-mediated inhibition of epidermal growth factor receptor gene expression using short interfering nucleic acids
INVENTOR(S): McSwiggen, James A.
PATENT ASSIGNEE(S): USA

SOURCE: U.S. Pat. Appl. Publ., 307 pp., Cont.-in-part of U.S. Ser. No. 163,552.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 233
PATENT INFORMATION:

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EP 1501853	A2	20050202	EP 2003-716093	20030220
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JP 2005517437	T2	20050616	JP 2003-569805	20030220
US 2005176024	A1	20050811	US 2004-923354	20040820

PRIORITY APPLN. INFO.:

US 2001-296249P	P	20010606
US 2001-916466	A2	20010725
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US 2002-163552	A2	20020606
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AU 1995-26422	A3	19950518
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US 1997-36476P	P	19970131
US 1997-985162	A1	19971204
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US 2001-311865P	P	20010813
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WO 2002-US15876	A2	20020520
WO 2002-US16840	A1	20020529
US 2002-386782P	P	20020606
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US 2002-408378P	P	20020905
US 2002-409293P	P	20020909
US 2002-251117	A1	20020919
US 2002-277494	A1	20021021
US 2003-440129P	P	20030115
WO 2003-US5028	A2	20030220
WO 2003-US5045	W	20030220
WO 2003-US5346	A2	20030220

US 2003-427160	A2	20030430
US 2003-444853	A2	20030523
US 2003-693059	A2	20031023
US 2003-720448	A2	20031124
US 2003-724270	A2	20031126
US 2003-727780	A2	20031203
US 2004-757803	A2	20040114
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US 2004-780447	A2	20040213
US 2004-826966	A2	20040416
WO 2004-US13456	A2	20040430
WO 2004-US16390	A2	20040524

OTHER SOURCE(S): MARPAT 139:240335

AB The present invention concerns methods and reagents useful in modulating epidermal growth factor receptor gene (HER1, **HER2** also known as **erbB2/neu**, HER3, and HER4) expression in a variety of applications, including use in therapeutic, diagnostic, target validation, and genomic discovery applications. Specifically, the invention relates to small nucleic acid mols., such as short interfering nucleic acid (siNA), short interfering RNA (**siRNA**), double-stranded RNA (**dsRNA**), micro-RNA (miRNA), and short hairpin RNA (shRNA) mols. capable of mediating RNA interference (**RNAi**) against epidermal growth factor receptor gene expression and/or activity. Exemplary siNA mols. are synthesized in tandem using standard phosphoramidite synthesis chemical and a cleavable linker, for example a succinyl-based linker, followed by a one-step purification process that provides **RNAi** mols. in high yield. Chemical modifications (2'-O-Me and 2'-deoxy-2'-fluoro groups, phosphorothioate linkages, 5'-terminal caps comprising an inverted deoxy abasic moiety, etc.) in siNA constructs are selected to yield nuclease resistance while preserving the ability to mediate **RNAi** activity. The siNA mols. are designed that can bind to each target and are optionally individually analyzed by a computer folding algorithm to assess whether the siNA mol. can interact with the target sequence. The efficacy of siNAs targeting epidermal growth factor receptor genes are tested in cell culture using, for example, SKBR-3 or SKOV-3 cells, to detn the extent of RNA and protein inhibition; after an optimal transfection agent concentration is chosen, a RNA time-course of inhibition is performed with the lead siNA mol(s). The siNA mols. are useful in the treatment and diagnosis of cancer.

L5 ANSWER 8 OF 14 CA COPYRIGHT 2006 ACS on STN DUPLICATE 1

ACCESSION NUMBER: 138:19530 CA

TITLE: Nucleic acid treatment of diseases or conditions related to levels of Ras, HER2 and HIV

INVENTOR(S): McSwiggen, James

PATENT ASSIGNEE(S): Ribozyme Pharmaceuticals, Incorporated, USA

SOURCE: PCT Int. Appl., 185 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 233

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002097114	A2	20021205	WO 2002-US16840	20020529
WO 2002097114	A3	20030508		

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WO 2003070912	A3	20041111		

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JP 2005517437	T2	20050616	JP 2003-569805	20030220
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US 2005176024	A1	20050811	US 2004-923354	20040820
US 2005288242	A1	20051229	US 2004-923476	20040820

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US 2001-294140P	P	20010529
US 2001-296249P	P	20010606
US 2001-318471P	P	20010910
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US 1996-623891	A	19960325
AU 1996-76662	A3	19961025
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US 2002-409293P	P	20020909
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US 2002-277494	A1	20021021
US 2003-440129P	P	20030115
WO 2003-US5028	A2	20030220
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US 2004-543480P	P	20040210
US 2004-780447	A2	20040213
US 2004-826966	A2	20040416
WO 2004-US13456	A2	20040430
WO 2004-US16390	A2	20040524

AB The present invention relates to nucleic acid mols., including enzymic nucleic acid mols., such as DNAzymes (e.g. DNA enzymes, catalytic DNA), siRNA, aptamers, and antisense that modulate the expression of Ras genes such as K-Ras, H-Ras, and/or N-Ras, HIV genes such as HIV-1, and HER2 (c-erbB2) gene. The sequence of human HER2 or Ras genes were screened for accessible sites using a computer-folding algorithm. Regions of the RNA that do not form secondary folding structure and contain potential enzymic nucleic acid mol. and/or antisense binding/cleavage sites are identified. The sequences of c-Ki-ras, c-Ha-ras, HER2, and HIV RNA binding/cleavage sites are provided, as are the sequences of designed enzymic nucleic acid mols., e.g., hammerhead ribozymes, DNAzymes, inozymes, zinzymes, and Amberzymes. [This abstract record is one of two records for this document necessitated by the large number of index entries required to fully index the document and publication system constraints.].

L5 ANSWER 9 OF 14 CA COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 137:104780 CA
TITLE: Method for inhibiting the expression of a target gene
INVENTOR(S): Kreutzer, Roland; Limmer, Stephan; Rost, Sylvia; Hadwiger, Philipp
PATENT ASSIGNEE(S): Ribopharma Ag, Germany
SOURCE: PCT Int. Appl., 203 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 11
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002055693	A2	20020718	WO 2002-EP152	20020109 <--
WO 2002055693	A3	20030717		

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DE 10100586 C1 20020411 DE 2001-10100586 20010109 <--

DE 10160151 A1 20030626 DE 2001-10160151 20011207

CA 2432350 AA 20020718 CA 2002-2432350 20020109 <--

EP 1352061 A2 20031015 EP 2002-710786 20020109

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JP 2004519458 T2 20040702 JP 2002-556740 20020109

CN 1630724 A 20050622 CN 2002-803557 20020109

DE 10230996 A1 20030717 DE 2002-10230996 20020709

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WO 2003033700 A1 20030424 WO 2002-EP11432 20021011

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WO 2003035868 A1 20030501 WO 2002-EP11968 20021025

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WO 2003035869 A1 20030501 WO 2002-EP11969 20021025

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WO 2003035870 A1 20030501 WO 2002-EP11970 20021025

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WO 2003035082 A1 20030501 WO 2002-EP11971 20021025

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			DE 2002-10230997	A	20020709
			DE 2002-10235620	A	20020802
			DE 2002-10235621	A	20020802
			WO 2002-EP11432	A2	20021011
			WO 2002-EP11968	A2	20021025
			WO 2002-EP11969	W	20021025
			WO 2002-EP11970	W	20021025
			WO 2002-EP11971	A2	20021025
			WO 2002-EP11972	W	20021025

WO 2002-EP11973 W 20021025

AB A method of inhibiting the expression of a specific gene using double-stranded oligoribonucleotides (**dsRNA**) capable of hybridizing with the gene is described. The **dsRNA** has a double-stranded core that is no more than 49 base-pairs long and has one or two 1-4 nucleotide single-stranded ends. Inhibition of expression of green fluorescent protein reporter gene in vitro and in vivo (3T3 and HeLa-S3 cells) by double-stranded RNA was demonstrated. A 21-nucleotide oligoribonucleotide, to which the complementary oligoribonucleotide was covalently attached via a linker, was shown to be an effective inhibitor. Inhibition of expression of the genes for epidermal growth factor receptors in the glioblastoma cell line U87MG is demonstrated.

L5 ANSWER 10 OF 14 CA COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 137:104740 CA
TITLE: Targeted inhibition of gene expression with double-stranded RNA with single-stranded ends
INVENTOR(S): Kreutzer, Roland; Limmer, Stefan; Rost, Sylvia; Hadwiger, Philipp
PATENT ASSIGNEE(S): Ribopharma A.-G., Germany
SOURCE: Ger. Offen., 100 pp.
CODEN: GWXXBX
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 10100588	A1	20020718	DE 2001-10100588	20010109 <--

PRIORITY APPLN. INFO.: DE 2001-10100588 20010109

AB A method of inhibiting the expression of a specific gene using double-stranded oligoribonucleotides (**dsRNA**) capable of hybridizing with the gene is described. The **dsRNA** has a double-stranded core that is no more than 49 base-pairs long and has 1-4 nucleotide single-stranded ends. The RNA may be delivered as single-stranded RNAs that hybridize to create the duplex, e.g. as nucleotide-resistant phosphorothioate oligonucleotides. The RNA may be made up of three oligonucleotides with the overlaps between the individual pairs of oligonucleotides being no more than 25 base pairs. This structure ensures that the ends are in the same orientation with respect to the target sequence. Inhibition of expression of green fluorescent protein reporter gene in vitro and in vivo (3T3 cells) by double-stranded RNA was demonstrated. A 21-nucleotide oligoribonucleotide, to which the complementary oligoribonucleotide was covalently attached via a linker, was shown to be an effective inhibitor.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 11 OF 14 CA COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 137:380906 CA
TITLE: Inhibition of gene expression with double-stranded oligoribonucleotides in interferon-treated cells
INVENTOR(S): Kreutzer, Roland; Limmer, Stefan; Rost, Sylvia; Hadwiger, Philipp
PATENT ASSIGNEE(S): Ribopharma AG, Germany
SOURCE: Ger., 98 pp.
CODEN: GWXXAW
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

DE 10100587	C1	20021121	DE 2001-10100587	20010109 <--

PRIORITY APPLN. INFO.: DE 2001-10100587 20010109

AB The invention concerns a method for inhibiting expression of a target gene in a cell comprising introducing at least one double-stranded oligoribonucleotide (**dsRNA**) of no more than 49 bp in a sufficient quantity for the inhibition of the expression of the target gene. Preferably, at least one end of the **dsRNA** contains non-Watson-Crick-paired nucleotides, or consists of 1-4 unpaired nucleotides. One strand, or a portion of one strand, of the **dsRNA** is complementary to the target gene. The cell is treated with interferon before induction of the **dsRNA**.

REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 12 OF 14 CA COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 136:305087 CA

TITLE: Targeted inhibition of gene expression with double-stranded RNA with single-stranded ends

INVENTOR(S): Kreutzer, Roland; Limmer, Stefan; Rost, Sylvia; Hadwiger, Philipp

PATENT ASSIGNEE(S): Ribopharma A.-G., Germany

SOURCE: Ger., 104 pp.
CODEN: GWXXAW

DOCUMENT TYPE: Patent

LANGUAGE: German

FAMILY ACC. NUM. COUNT: 11

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 10100586	C1	20020411	DE 2001-10100586	20010109 <--
CA 2432341	AA	20020718	CA 2002-2432341	20020109 <--
CA 2432350	AA	20020718	CA 2002-2432350	20020109 <--
WO 2002055692	A2	20020718	WO 2002-EP151	20020109 <--
WO 2002055692	A3	20030612		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
WO 2002055693	A2	20020718	WO 2002-EP152	20020109 <--
WO 2002055693	A3	20030717		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1349927	A2	20031008	EP 2002-702247	20020109
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
EP 1352061	A2	20031015	EP 2002-710786	20020109
EP 1352061	B1	20060531		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,				

IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

JP 2004519457	T2	20040702	JP 2002-556739	20020109
JP 2004519458	T2	20040702	JP 2002-556740	20020109
CN 1630724	A	20050622	CN 2002-803557	20020109
CN 1650010	A	20050803	CN 2002-803555	20020109
US 2004001811	A1	20040101	US 2003-384260	20030307
US 2004175703	A1	20040909	US 2003-384339	20030307
ZA 2003004127	A	20040511	ZA 2003-4127	20030528
ZA 2003004500	A	20031118	ZA 2003-4500	20030610
US 2005176667	A1	20050811	US 2004-941663	20040915
US 2006084621	A1	20060420	US 2005-229183	20050915
PRIORITY APPLN. INFO.:			DE 2001-10100586	A 20010109
			DE 2001-10155280	A 20011026
			DE 2001-10158411	A 20011129
			DE 2001-10160151	A 20011207
			WO 2002-EP151	W 20020109
			WO 2002-EP152	W 20020109
			US 2003-384260	A2 20030307
			US 2004-941663	A2 20040915

AB A method of inhibiting the expression of a specific gene using double-stranded oligoribonucleotides (**dsRNA**) capable of hybridizing with the gene is described. The **dsRNA** has a double-stranded core that is no more than 49 base-pairs long and has 1-4 nucleotide single-stranded ends. Inhibition of expression of green fluorescent protein reporter gene in vitro and in vivo (3T3 cells) by double-stranded RNA was demonstrated. A 21-nucleotide oligoribonucleotide, to which the complementary oligoribonucleotide was covalently attached via a linker, was shown to be an effective inhibitor.

REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 13 OF 14 EMBASE

ACCESSION NUMBER: 2003064850 EMBASE

TITLE: The effectiveness of double-stranded short inhibitory RNAs (**siRNAs**) may depend on the method of transfection.

AUTHOR: Walters D.K.; Jelinek D.F.

CORPORATE SOURCE: Dr. D.F. Jelinek, 200 First Street, S. W., Rochester, MN 55905, United States. jelinek.diane@mayo.edu

SOURCE: Antisense and Nucleic Acid Drug Development, (2002) Vol. 12, No. 6, pp. 411-418. .
Refs: 17
ISSN: 1087-2906 CODEN: ANADF5

COUNTRY: United States

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 016 Cancer
022 Human Genetics
037 Drug Literature Index

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 20 Feb 2003
Last Updated on STN: 20 Feb 2003

AB RNA interference (**RNAi**) is a recently described powerful experimental tool that can cause sequence-specific gene silencing, thereby facilitating functional analysis of gene function. Consequently, we became interested in using **RNAi** to determine the function of aberrantly expressed ErbB3 in the KAS-6/1 human myeloma cell line. Despite the wealth of information available on the use of **RNAi**, **dsRNA** target design, and the transfection of **dsRNA** in vitro, little information is available for transfecting **dsRNA** into nonadherent cells from any species. In the present study, we report that gene silencing of ErbB3 was not observed in myeloma cells when **dsRNA** targeting ErbB3 was introduced using conventional

transfection agents and protocols that have proved successful for several adherent cell lines. Silencing of ErbB3, however, was observed in T47D cells, an adherent breast carcinoma cell line, using the same transfection methods, indicating that our target sequence was functional for gene silencing of ErbB3. Interestingly, ErbB3 was silenced in myeloma cells when the **dsRNA** target was introduced by electroporation. Thus, our studies illustrate the striking dependence of **dsRNA**-mediated gene silencing in some cells on the methods of **dsRNA** transfection.

L5 ANSWER 14 OF 14 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 91347216 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1715233
TITLE: A distinct kinase modulates the expression of IFN-inducible genes in human breast cancer cells.
AUTHOR: Tiwari R K; Osborne M P
CORPORATE SOURCE: Breast Cancer Research Laboratory, Memorial Sloan Kettering Cancer Center, New York, N.Y. 10021.
CONTRACT NUMBER: P-01 CA29502 (NCI)
SOURCE: Cancer letters, **(1991 Jul 26)** Vol. 59, No. 1, pp. 31-6.
Journal code: 7600053. ISSN: 0304-3835.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199110
ENTRY DATE: Entered STN: 20 Oct 1991
Last Updated on STN: 3 Mar 2000
Entered Medline: 2 Oct 1991

AB The biological activity of interferons (IFNs) is presumed to be mediated through the induction of a number of IFN-inducible genes. IFN-mediated gene induction was examined in two human breast cancer cell lines, MCF-7 and BT-20. Both these cell lines were remarkably responsive to IFNs as a number of IFN inducible genes were rapidly induced. We examined the sensitivity of these genes towards 2-aminopurine (2-AP), a known inhibitor of double-stranded (ds) RNA dependent protein kinase. 2-AP has also been reported to inhibit the induction of IFN-beta 1 in response to **dsRNA** and the genes c-myc and c-fos in fibroblasts. In both MCF-7 and BT-20 cell lines, 2-AP selectively inhibited the IFN-induced gene responses. 2-AP did not affect levels of the oncogene, HER-2/**neu** . Tamoxifen (TAM), an antiestrogenic drug, which is known to inhibit the activity of protein kinase C at high concentrations, did not affect IFN-mediated gene induction. Our data is consistent with the concept that the 2-AP sensitive kinase is primarily associated with the IFN-induced gene systems and that positive and negative growth regulating stimuli in breast cancer may require the participation of distinct kinases.